

L Number	Hits	Search Text	DB	Time stamp
2	2	9851331.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:34
7	2	6123916.pn.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 06:52
8	2	6123916.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 06:52
5	2	4853371.pn. and somatostatin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
9	0	4853371.pn. and somatostatin and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
10	0	4853371.pn. and somatostatin and fat	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:07
11	2	4853371.pn. and somatostatin and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
12	0	4853371.pn. and somatostatin and insulin and weight	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
13	0	4853371.pn. and somatostatin and insulin and calorie	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
14	0	4853371.pn. and somatostatin and insulin and caloric	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
15	0	4853371.pn. and somatostatin and insulin and food	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:08
16	0	4853371.pn. and somatostatin and insulin and intake	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:09
17	3539	somatostatin and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:35
18	582	somatostatin and insulin and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:35
20	1	somatostatin.clm. and insulin.clm. and obesity.clm. and hypersecretion	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:35
21	21	somatostatin and insulin and obesity and hypersecretion	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:36
22	7	somatostatin and insulin SAME hypersecretion and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:37

Search History 10/1/03 7:40:50 AM Page 1

c:\APPS\EAST\Workspaces\10006738.wsp

Copied from 10067738 on 22-04-2004

23	3	somatostatin.clm. and insulin SAME hypersecretion and obesity	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:37
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25	1	somatostatin.clm. and insulin SAME hypersecretion and obesity.clm. and insulin.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:38
19	20	somatostatin.clm. and insulin.clm. and obesity.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:39
26	12	obesity.ab. and somatostatin.clm. and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:39
27	10	obesity.ab. and somatostatin.clm. and insulin.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:40
28	0	514/2.ccls. and obesity.ab. and somatostatin.clm. and insulin.clm.	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:40
29	22	514/2.ccls. and somatostatin and obesity and insulin	USPAT; US-PGPUB; EPO; JPO; DERWENT	2003/10/01 07:40

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B 155, 73, 344, 358, 35, 65

01oct03 07:04:45 User268152 Session D29.1

\$0.00 0.134 DialUnits FileHomeBase

\$0.00 Estimated cost FileHomeBase

\$0.17 INTERNET

\$0.17 Estimated cost this search

\$0.17 Estimated total session cost 0.134 DialUnits

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File 155:MEDLINE(R) 1966-2003/Sep W4

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File 73:EMBASE 1974-2003/Sep W3

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File 344:Chinese Patents Abs Aug 1985-2003/Apr

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File 358:Current BioTech Abs 1983-2003/Aug

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File 65:Inside Conferences 1993-2003/Sep W4

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Set	Items	Description
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S AU=((LUSTIG, R?) OR (LUSTIG R?))

10 AU=LUSTIG, R?

129 AU=LUSTIG R?

S1 139 AU=((LUSTIG, R?) OR (LUSTIG R?))

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S S1 AND SOMATOSTATIN

139 S1

45966 SOMATOSTATIN

S2 5 S1 AND SOMATOSTATIN

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&gt;&gt;&gt;'1' invalid after set or accession number

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&gt;&gt;&gt;'1' invalid after set or accession number

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Display 2/2/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

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11919034 99362531 PMID: 10431109

**Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by a somatostatin agonist.**

Lustig R H; Rose S R; Burghen G A; Velasquez-Mieyer P; Broome D C; Smith K; Li H; Hudson M M; Heideman R L; Kun L E

Department of Pediatrics, University of Tennessee, Memphis, USA.

Journal of pediatrics (UNITED STATES) Aug 1999, 135 (2 Pt 1) p162-8,

ISSN 0022-3476 Journal Code: 0375410

Contract/Grant No.: M01-RR00211; RR; NCRR

Comment in J Pediatr. 1999 Aug;135(2 Pt 1) 142-4; Comment in PMID 10431105

Document type: Clinical Trial; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

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>>>'1' invalid after set or accession number

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T FULL/S2/1-5

>>>'FULL' not recognized as set or accession number

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T S2/1-5

>>>'-' not allowed as format type

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T 1-5

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T S2

2/2/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

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Document type: Clinical Trial; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: AIM; INDEX MEDICUS

Tags: Animal; Female; Human; Male; Support, U.S. Gov't, P.H.S.

Descriptors: \*Brain Damage, Chronic--complications--CO; \*Hormones, Synthetic--therapeutic use--TU; \*Hypothalamic Diseases--drug therapy--DT; \*Obesity--drug therapy--DT; \*Octreotide--therapeutic use--TU; \*Somatostatin--agonists--AG; Adolescent; Child; Disease Models, Animal; Hyperphagia--drug therapy--DT; Hyperphagia--etiology--ET; Hyperphagia--physiopathology--PP; Hypothalamic Diseases--etiology--ET; Hypothalamic Diseases

--physiopathology--PP; Insulin--blood--BL; Obesity--etiology--ET; Obesity  
--physiopathology--PP; Rats  
CAS Registry No.: 0 (Hormones, Synthetic); 11061-68-0 (Insulin);  
51110-01-1 (Somatostatin); 83150-76-9 (Octreotide)  
Record Date Created: 19990824  
Record Date Completed: 19990824

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Display 2/2/2 (Item 2 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

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10110295 22074690 PMID: 12079271

Use of somatostatin receptor ligands in obesity and diabetic complications.

Boehm Bernhard O; Lustig Robert H  
Division of Endocrinology, Ulm University, Robert-Koch-Strasse 8,  
Ulm/Donau, 89070, Germany.

Best practice & research. Clinical gastroenterology (England) Jun 2002,  
16 (3) p493-509, ISSN 1521-6918 Journal Code: 101120605

Document type: Journal Article; Review; Review Literature

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Subfile: INDEX MEDICUS

Tags: Human

Descriptors: \*Diabetic Retinopathy--drug therapy--DT; \*Obesity --drug

-more-

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T S2/6/1-5

2/6/1 (Item 1 from file: 155)

11919034 99362531 PMID: 10431109

Hypothalamic obesity caused by cranial insult in children: altered  
glucose and insulin dynamics and reversal by a somatostatin agonist.

Aug 1999

2/6/2 (Item 2 from file: 155)

10110295 22074690 PMID: 12079271

Use of somatostatin receptor ligands in obesity and diabetic complications.

Jun 2002

2/6/3 (Item 1 from file: 73)

11958336 EMBASE No: 2003069647

Autonomic dysfunction of the beta-cell and the pathogenesis of obesity  
2003

2/6/4 (Item 2 from file: 73)

11631097 EMBASE No: 2002202317

Hypothalamic obesity: The sixth cranial endocrinopathy  
2002

2/6/5 (Item 3 from file: 73)

10654494 EMBASE No: 2000120584

Hypothalamic obesity caused by cranial insult in children: Altered  
glucose and insulin dynamics and reversal by a somatostatin agonist  
1999

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T S2/3,AB/1-5

>>>No matching display code(s) found in file(s): 65

**2/3,AB/1 (Item 1 from file: 155)**

DIALOG(R) File 155:MEDLINE(R)

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11919034 99362531 PMID: 10431109

**Hypothalamic obesity caused by cranial insult in children: altered glucose and insulin dynamics and reversal by a somatostatin agonist.**

Lustig R H; Rose S R; Burghen G A; Velasquez-Mieyer P; Broome D C; Smith K; Li H; Hudson M M; Heideman R L; Kun L E

Department of Pediatrics, University of Tennessee, Memphis, USA.

Journal of pediatrics (UNITED STATES) Aug 1999, 135 (2 Pt 1) p162-8, ISSN 0022-3476 Journal Code: 0375410

Contract/Grant No.: M01-RR00211; RR; NCRR

Comment in J Pediatr. 1999 Aug;135(2 Pt 1) 142-4; Comment in PMID 10431105

Document type: Clinical Trial; Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

**OBJECTIVE:** Hypothalamic obesity is a rare sequela of cranial insult, for which pathogenesis and treatment remain obscure. In rodents ventromedial hypothalamic damage causes hyperphagia, obesity, hyperinsulinism, and insulin resistance. Reduction of insulin secretion in humans may attenuate weight gain. **METHODS:** Eight children with intractable obesity after therapy for leukemia or brain tumors underwent oral glucose tolerance testing (OGTT) with simultaneous insulin levels before and after treatment with octreotide for 6 months. **RESULTS:** In comparison with a 6-month pre-study observation period, patients exhibited weight loss (+6.0 +/- 0.7 kg vs -4.8 +/- 1.8 kg; P = .04) and decrease in body mass index (+2.1 +/- 0.3 kg/m(2) vs -2.0 +/- 0.7 kg/m(2); P = .0001). Recall calorie count decreased during the 6 months of treatment (P = .015). OGTT demonstrated biochemical glucose intolerance in 5 of 8 patients initially and in 2 of 7 at study end, whereas insulin response was decreased (281 +/- 47 microU/mL vs 114 +/- 35 microU/mL; P = .04). Percent weight change correlated with changes in insulin response (r = 0.72, P = .012) and changes in plasma leptin r = 0.76, P = .0004). **CONCLUSIONS:** Patients with hypothalamic obesity demonstrate excessive insulin secretion. Octreotide administration promoted weight loss, which correlated with reduction in insulin secretion on OGTT and with reduction in leptin levels. Pre-study biochemical glucose tolerance improved in several patients while they were receiving octreotide. These results suggest that normalization of insulin secretion may be an effective therapeutic strategy in this syndrome.

**2/3,AB/2 (Item 2 from file: 155)**

DIALOG(R) File 155:MEDLINE(R)

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10110295 22074690 PMID: 12079271

**Use of somatostatin receptor ligands in obesity and diabetic complications.**

Boehm Bernhard O; Lustig Robert H

Division of Endocrinology, Ulm University, Robert-Koch-Strasse 8, Ulm/Donau, 89070, Germany.

Best practice & research. Clinical gastroenterology (England)

16 (3) p493-509, ISSN 1521-6918 Journal Code: 101120605

Document type: Journal Article; Review; Review Literature

Languages: ENGLISH

Main Citation Owner: NLM

Record type: Completed

Somatostatin (SMS) is a potent inhibitory molecule. It inhibits both exocrine and endocrine secretory functions of the pancreas, suppresses

Jun 2002,

growth hormone secretion and reduces the level of insulin-like growth factor-1. Long-acting somatostatin analogues were currently investigated for potential clinical benefits in two settings: (a) control of hyperinsulinaemia in obesity and (b) control of an excess of pro-angiogenic factors in diabetes-associated retinal complications. In two randomized, controlled trials the long-acting somatostatin analogue octreotide retarded progression of the microvascular complications in pre-proliferative and advanced stages of diabetic retinopathy. Inhibition of the early phase of insulin secretion by use of octreotide in patients with hypothalamic obesity resulted in weight loss and improved quality of life. Efficacy of octreotide correlated to residual beta-cell activity prior to the treatment. Obesity and diabetes mellitus are the most common chronic metabolic disorders in the world. The use of somatostatin analogues addressing the various hormonal imbalances of these disorders may provide a novel concept for their pharmacological treatment. Copyright 2002 Elsevier Science Ltd.

**2/3,AB/3 (Item 1 from file: 73)**

DIALOG(R) File 73:EMBASE

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11958336 EMBASE No: 2003069647

**Autonomic dysfunction of the beta-cell and the pathogenesis of obesity**

Lustig R.H.

Prof. R.H. Lustig, Department of Clinical Pediatrics, Division of Endocrinology, University of California, San Francisco, CA 94143-0136 United States

AUTHOR EMAIL: [rlustig@peds.ucsf.edu](mailto:rlustig@peds.ucsf.edu)

Reviews in Endocrine and Metabolic Disorders ( REV. ENDOCR. METAB. DISORD. ) (Netherlands) (2003) 4/1 (23-32)

CODEN: REMDC ISSN: 1389-9155

DOCUMENT TYPE: Journal ; Review

LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH

NUMBER OF REFERENCES: 76

In this review, we describe and characterize a specific subtype of obesity with organic underpinnings. There is evidence for etiology (vagal modulation of beta-cell depolarization), pathogenesis (insulin hypersecretion), diagnosis (insulin dynamics during OGTT), and treatment (insulin suppression through beta-cell somatostatin receptor agonism). Although the number of obese patients with organic VMH damage is exceedingly small, the numbers of subjects who may manifest similar pathogenesises, with either a genetic, neural, or hormonal etiology, may be much greater. Studies are now underway to determine the incidence of this disorder, and the best method for diagnosis and treatment. This recognition of this syndrome of autonomic dysfunction of beta-cell insulin secretion is an important first step in improving the nosology of obesity, tying the hypothalamus to the adipocyte, and trying to correlate biochemistry with human behavior. In doing so, it is anticipated that the clinical evaluation of obesity will take a more scientific tone in the near future.

**2/3,AB/4 (Item 2 from file: 73)**

DIALOG(R) File 73:EMBASE

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11631097 EMBASE No: 2002202317

**Hypothalamic obesity: The sixth cranial endocrinopathy**

Lustig R.H.

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AUTHOR EMAIL: [rlustig@peds.ucsf.edu](mailto:rlustig@peds.ucsf.edu)

Endocrinologist ( ENDOCRINOLOGIST ) (United States) 2002, 12/3  
(210-217)  
CODEN: EDOCE ISSN: 1051-2144  
DOCUMENT TYPE: Journal ; Review  
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH  
NUMBER OF REFERENCES: 70

The hypothalamus is "ground zero" for the neuroendocrine control of five hormonal systems, which are mediated through negative feedback regulation of pituitary hormone release. Energy balance is regulated by a more complex neuroendocrine feedback loop. The hypothalamus integrates peripheral neural and hormonal afferent signals of satiety and energy reserve and directs neuroendocrine efferent arms to effect energy storage versus expenditure; however, in this feedback loop, the pituitary is not integral. Damage to this hypothalamic control system results in a syndrome of intractable weight gain. This syndrome of hypothalamic obesity is usually caused by cranial insult, such as brain trauma, tumor, surgery, or radiation. In some cases, however, it may have a congenital cause. The cause and pathogenesis of obesity in such subjects is akin to an animal model of obesity in which the ventromedial hypothalamus (VMH) is destroyed or deafferented. The VMH-lesioned rat exhibits a vagally mediated potentiation of insulin secretion in response to glucose. Excess insulin secretion favors and promotes partitioning of energy substrate into fat, even with caloric restriction. Similarly, patients with hypothalamic obesity exhibit insulin hypersecretion. By suppressing insulin release at the beta cell in a specific manner using the somatostatin agonist octreotide, the shunting of energy substrate to adipose is attenuated. Treated patients exhibit weight loss and improved quality of life, which correlate with insulin suppression. Thus, hypothalamic obesity is the sixth cranial endocrinopathy, with an identifiable cause, pathogenesis, diagnosis, and treatment.

2/3,AB/5 (Item 3 from file: 73)  
DIALOG(R)File 73:EMBASE  
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10654494 EMBASE No: 2000120584

**Hypothalamic obesity caused by cranial insult in children: Altered glucose and insulin dynamics and reversal by a somatostatin agonist**  
Lustig R.H.; Rose S.R.; Burghen G.A.; Velasquez-Mieyer P.; Broome D.C.; Smith K.; Li H.; Hudson M.M.; Heideman R.L.; Kun L.E.  
Dr. R.H. Lustig, Department of Pediatrics, Methodist LeBonheur Child. Med. Ctr., 50 North Dunlap, Memphis, TN 38103 United States  
Journal of Pediatrics ( J. PEDIATR. ) (United States) 1999, 135/2 I (162-168)  
CODEN: JOPDA ISSN: 0022-3476  
DOCUMENT TYPE: Journal; Article  
LANGUAGE: ENGLISH SUMMARY LANGUAGE: ENGLISH  
NUMBER OF REFERENCES: 42

Objective: Hypothalamic obesity is a rare sequela of cranial insult, for which pathogenesis and treatment remain obscure. In rodents ventromedial hypothalamic damage causes hyperphagia, obesity, hyperinsulinism, and insulin resistance. Reduction of insulin secretion in humans may attenuate weight gain. Methods: Eight children with intractable obesity after therapy for leukemia or brain tumors underwent oral glucose tolerance testing (OGTT) with simultaneous insulin levels before and after treatment with octreotide for 6 months. Results: In comparison with a 6-month pre-study observation period, patients exhibited weight loss (+6.0 +/- 0.7 kg vs -4.8 +/- 1.8 kg; P = .04) and decrease in body mass index (+2.1 +/- 0.3 kg/msup 2 vs -2.0 +/- 0.7 kg/msup 2; P = .0001). Recall calorie count decreased during the 6 months of treatment (P = .015). OGTT demonstrated biochemical glucose intolerance in 5 of 8 patients initially and in 2 of 7 at study



end, whereas insulin response was decreased ( $281 \pm 47$   $\mu$ U/mL vs  $114 \pm 35$   $\mu$ U/mL;  $P = .04$ ). Percent weight change correlated with changes in insulin response ( $r = 0.72$ ,  $P = .012$ ) and changes in plasma leptin  $r = 0.76$ ,  $P = .0004$ ). Conclusions: Patients with hypothalamic obesity demonstrate excessive insulin secretion. Octreotide administration promoted weight loss, which correlated with reduction in insulin secretion on OGTT and with reduction in leptin levels. Pre-study biochemical glucose tolerance improved in several patients while they were receiving octreotide. These results suggest that normalization of insulin secretion may be an effective therapeutic strategy in this syndrome.

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